Respiratory system, lung, and chest wall mechanics after longitudinal laparotomy in rats

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ABSTRACT: It has been demonstrated that respiratory resistance and elastance increase whilst the abdomen remains open during longitudinal laparotomy. We wished to determine whether changes also occur after abdominal closure in the same animal preparation.

In 10 sedated, anaesthetized paralysed, and mechanically-ventilated rats (309±33 (sd) g), resistances and elastances of the respiratory system, lung, and chest wall were measured both before longitudinal laparotomy and directly after abdominal closure. Furthermore, the resistances were also split into their initial and difference components, the former reflecting the Newtonian resistances and the latter representing the viscoelastic/inhomogeneous pressure dissipations in the system. For this purpose, the end inflation occlusion during constant inspiratory flow method was used.

After laparotomy, no statistically significant changes were found in elastances and resistances of the respiratory system, lungs and chest wall (paired Student’s t-test, significance level=5%). It can be concluded that after midline xiphipubic laparotomy accompanied by bilateral ventro-dorsal infracostal incision, respiratory resistances and elastances were not different from those found in the control condition.

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Laparotomy often leads to respiratory impairment postoperatively. Several potential mechanisms may be suggested to explain muscular dysfunction after this surgery [1–3]. However, postoperative respiratory mechanical changes are not well understood. It has recently been demonstrated in guinea-pigs and rats [4, 5] that, during longitudinal laparotomy, respiratory system elastance increase because of its lung and chest wall components. Respiratory system resistance was also found to increase whilst the abdomen remained open [4, 5], as a result higher mechanical inhomogeneities and/or viscoelastic properties of the chest wall [4]. Furthermore, although pulmonary contribution to respiratory system inhomogeneities/viscoelasticity is quite well understood [6–8], the relative contributions of the rib cage and abdomen components to those properties have only recently received much attention [6–13]. Hence, in the present investigation, the previous studies using the laparotomy model [4, 5] have been extended to verify the hypothesis that closure of the laparotomic cut could normalize lung and chest wall mechanics.

Methods

Ten adult Wistar rats (weight 230–355 g mean 309±33 (sd)) g were initially sedated with diazepam (5 mg i.p.), and anaesthetized with pentobarbital sodium (20 mg kg⁻¹ i.p.). A snugly fitting cannula (1.5 mm internal diameter (ID)) was introduced into the trachea under direct vision aided by a small laryngoscope. The rats were then placed in the supine position on a heated surgical table.

An adequate pneumotachograph [14] was connected to the tracheal cannula for the measurements of airflow (V) and, by electronic integration, of tidal volume (V). The flow resistance of the equipment (tracheal cannula included) (Req), was constant up to flow rates of 26 ml·s⁻¹ and amounted to 0.03 cmH₂O·ml⁻¹·s. Because abrupt changes of diameter were not present in our circuit, errors of measurement of flow resistance were avoided [15, 16]. The equipment dead space was 0.4 ml. Tracheal pressure (Ptr) was measured with a Hewlett Packard 270 differential pressure transducer. Changes in oesophageal pressure (Poes) were measured with a 30 cm long water-filled catheter (PE-240), with side holes at the tip connected to a PR23-2D-300 Statham differential pressure transducer. The catheter was passed into the stomach and then slowly returned into the oesophagus; its proper positioning was assessed using the “occlusion test” [17]. The Ptr signal was then connected to the other port of the transducer so that changes in transpulmonary pressure (P=Ptr-Poes) were, thereafter, registered.

The rats were then paralysed with succinylcholine
chloride (5 mg·kg i.p.) and artificially ventilated with a constant flow ventilator. During the measurement periods, an end-inspiratory pause could be generated by adjusting the ventilator settings. In order to avoid the effects of different flows and tidal volumes, and thence inspiratory duration, on the measured variables [7, 8, 18], special care was taken to keep tidal volume (VT) and flow constant in all animals. The experiments lasted no more than 45 min.

The frequency responses of the pressure measurement systems (Ptr and Pl) were flat up to 20 Hz, without appreciable phase shift between the signals. All signals were conditioned and amplified in a Beckman type R Dynograph and recorded on paper at speeds of 1 and 5 mm·s⁻¹. The signals were also passed through 8 pole Bessel filters (902LPF, Frequency Devices) with their corner frequencies set at 100 Hz. They were then sampled at 200 Hz with a 12-bit analogue-to-digital converter (DT-2801A, Data Translation) and stored on computer. All data were collected using LABDAT software (RHT-InfoData).

The measurements were performed before incision and immediately after abdominal closure. The abdominal anterior wall was cut at the midline in the craniocaudal direction, and at the sides in the ventrodorsal direction caudad to the rib cage. In addition, in order to assess possible modifications in diaphragmatic resting position, the rats underwent radioscopic examination before incision and immediately after abdominal closure.

Respiratory mechanics were measured from end-inspiratory occlusions after constant flow inflations [19]. Although this method has been used for a long time, the significance of the measured variables has only recently been clarified [6, 8, 19–22]. After end-inspiratory occlusion, there is a fast initial drop in Ptr (ΔPinit,rs) from the preocclusion value up to an inflection point (Pl,rs) followed by a slower pressure drop (ΔPdiff,rs) to a plateau. This plateau corresponds to the elastic recoil pressure of the respiratory system (Pel,rs). ΔPinit,rs and ΔPdiff,rs divided by the flow immediately preceding the occlusion provide the initial, or viscous, (Rvis,rs) and difference (Rdiff,rs) resistances of the respiratory system, respectively. Rvis,rs selectively reflects the combination of airway and chest wall Newtonian resistances in normal animals [6, 18, 21]. Rdiff,rs, with the units of resistance, reflects stress relaxation of the lung and chest wall tissues, together with a tiny contribution of pendelluft in normal situations [6, 7, 23]. ΔPinit,rs + ΔPdiff,rs divided by flow gives the total respiratory system resistance (Rtot,rs). The same procedures apply to Pl, yielding the values of total (Rtot,w), initial (Rvis,w,), and difference (Rdiff,w) resistances of the lung. Total (Rtot,rs), initial (Rvis,rs, and difference (Rdiff,rs) resistances of the chest wall were calculated by subtracting the pulmonary from the corresponding respiratory system values. Req was subtracted from Rtot,rs, Rtot,rs, Rvis,rs, and Rvis,rs, so that the results reported here represent intrinsic resistance values. Respiratory system and lung elastances (Est,rs and Est,rs, respectively) were calculated by dividing the corresponding elastic recoil pressures (Pel,rs and Pel,rs, respectively) by VT. Chest wall elastance (Est,w) was calculated by subtracting Est,rs from Est,rs. Five to 10 determinations were performed in each animal, before and after surgery. Before each data collection period the airway contents were aspirated to remove possible mucus collection and the respiratory system was inflated to total lung capacity (Ptot=+30 cmH₂O). The manoeuvre was repeated three times.

Because a mechanical occlusion valve always takes a finite time to close, VT never drops to zero immediately upon interruption. The √ passing through the valve as it shuts increases the pressures, and may, thus, lead to an underestimation of Rtot and Rvis [7, 24]. The closing time of the valve used in the ventilator within the experimental range of inspiratory flows was 7 ms. The delay was allowed for by back-extrapolation of the pressure records to the actual time of occlusion and the corrections in resistance, although very minute, were performed as described previously [7].

All data were analysed using ANADAT data analysis software (RHT-InfoData).

Statistical analysis was performed by means of the Student’s paired t-test, with the significance level established at 5%.

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**Fig. 1.** – Respiratory system (Ers), lung (E L), and chest wall (Ew) elastances before surgery (Pre) and after abdominal closure (Post). Values are means ±/− SEM of seven rats. ○---○: total resistance (Rtot); Δ Δ: initial resistance (Rvis); ❏❏: difference resistance (Rdiff). In all instances, no statistically significant difference could be detected.

**Fig. 2.** – Respiratory system (Ers), lung (E L), and chest wall (Ew) static elastances before surgery (Pre) and after abdominal closure (Post). Values are means ±/− SEM of seven rats. In all instances, no statistically significant difference could be detected.
Results

Mean constant inspiratory flows (±SD) measured before and after surgery amounted to 8.96±0.46 and 9.18±0.61 ml·s⁻¹, respectively. The corresponding tidal volumes were 2.30±0.16 and 2.28±0.16 ml. No statistically significant difference could be detected.

Although a tendency to find higher respiratory system and lung resistances after abdominal closure could be noted (fig. 1), these changes were not statistically significant. The same applied to lung, chest wall, and respiratory system elastances (fig. 2).

Discussion

It is well known that, after laparotomy, respiratory dysfunction often occurs [25, 26], and pain [1], residual effects of anaesthetics and muscle relaxants [1], diaphragmatic fatigue [2], and neural reflexes [3] have been claimed to be responsible for it. Recently, studies in rats and guinea-pigs demonstrated that there are also mechanical factors leading to respiratory impairment while the abdomen remains open [4, 5]. However, the mechanical effects of longitudinal laparotomy have not so far been studied directly after abdominal closure.

As previously reported in rats [5] and guinea-pigs [4], the change in functional residual capacity (FRC) after abdominal opening was negligible, consisting of an expired volume not more than 0.1 ml. It could, thus, be assumed that in the present study no changes in FRC occurred, since the experiments were performed under similar conditions. Supporting this hypothesis, no difference in diaphragmatic resting position could be detected by radioscopic examination performed before and after surgery.

In spontaneously breathing dogs, it has been shown that, after longitudinal abdominal opening, there is a decrease in tidal volume accompanied by higher integrative inspiratory muscular electrical activity at the 2nd and 6th intercostal spaces [27]. It could be speculated that this finding is secondary to an increased mechanical impedance in the respiratory system. As already demonstrated in anaesthetized paralysed mechanically-ventilated rats [5], wide longitudinal laparotomy induced a significant increase in $R_{tot,rs}$ due to a rise in $R_{diff,rs}$. In a complementary study in guinea-pigs [4], $R_{diff,rs}$ was shown to increase following similar changes in $R_{diff,w}$. $R_{diff,w}$ can be closely related to stress relaxation or stress recovery properties of the chest wall tissues, together with a tiny contribution of asynchrony of movement within and between the chest wall components [2, 6, 23].

It is conceivable that after abdominal opening, changes in chest wall tissue viscoelastic properties are inclined to occur. In fact, the chest wall is made up of heterogeneous tissues: the rib cage consists primarily of the complex lever and joint system of the ribs, sternum, spinal column, and various muscle groups; the diaphragm-abdomen is composed of muscles of the abdominal wall, abdominal contents, and diaphragm. Some of these elements are modified by wide abdominal opening, whereas the remaining ones undergo a new pattern of movement from their relaxed configuration to tidal volume [5]. In the present investigation, no significant changes in respiratory resistance could be identified (fig. 1). It seems that the factor (or factors) leading to increased $R_{diff,w}$ whilst the abdomen remained open, was circumvented by abdominal closure.

In rodents, longitudinal abdominal incision increased $E_{st,rs}$ and $E_{st,w}$ [4, 5], diminished the lower rib cage circumference [5], and a cephalad diaphragmatic movement was observed [4, 5]. Thus, it can be concluded that the stretched diaphragm could have become less compliant. The configurational changes of the rib cage could also, possibly, account for an increased $E_{st,w}$. However, after abdominal closure, no statistically significant changes in elastance could be detected (fig. 2). It thus seems that abdominal wall suture restored chest wall mechanical integrity.

In conclusion, respiratory system, lung, and chest wall resistance and elastance remained unchanged after wide longitudinal laparotomy. These findings are important when considering the increased impedance of the system found whilst the abdomen remained open [4, 5, 27]. Moreover, they eliminate mechanical factors, as measured immediately after abdominal closure, as possible candidates for the respiratory impairment commonly found after wide laparotomy.

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References


